Physiology & Behavior 140 (2015) 203–208

Contents lists available at ScienceDirect

Physiology & Behavior

journal homepage: www.elsevier.com/locate/phb

# Sleep depth and continuity before and after chronic exercise in older men: Electrophysiological evidence

Michel O. Melancon [a,b], Dominique Lorrain [a,c,*], Isabelle J. Dionne [a,b]

[a] Research Center on Aging, Health and Social Services Centre—University Institute of Geriatrics of Sherbrooke, Sherbrooke, QC, Canada
[b] University of Sherbrooke, Faculty of Physical and Sport Education, Department of Kinanthropology, Sherbrooke, QC, Canada
[c] University of Sherbrooke, Faculty of Human Sciences, Department of Psychology, Sherbrooke, QC, Canada

## HIGHLIGHTS

- Older men showed more slow-wave sleep on active days following training.
- Wake time and REM onset latency were both reduced following exercise.
- Sleep continuity was improved due to reduced wake time with exercise.
- These effects of exercise on sleep, although modest, oppose those of aging.

## ARTICLE INFO

*Article history:*
Received 9 June 2014
Received in revised form 17 December 2014
Accepted 19 December 2014
Available online 22 December 2014

*Keywords:*
Aging
Chronic exercise
Physical activity
Deep sleep
Senior men
Sleep organization

## ABSTRACT

During later life sleep depth (slow-wave sleep, SWS) and maintenance exhibit deleterious changes, with possible negative effects on daytime function. This study assessed the effect of chronic, supervised exercise on sleep using laboratory-based polysomnography (PSG) and repeated measures in older adults. Thirteen men aged 64 ± 3 served as their own controls and had their sleep measured for a total of 6 nights: 3 before and 3 after the 16-week training intervention. Each sequence involved 1 familiarization trial followed by 2 experimental nights (exercise night; nonexercise night) measured using 13-channel PSG (combined electroencephalography, electromyography, and electro-oculography). The exercise challenges consisted of inclined treadmill brisk walking (60 min, 68–69% $\dot{V}O_2$ peak). The intervention successfully improved some parameters of aerobic fitness, i.e. ventilatory thresholds 1 and 2 ($P < 0.05$). Acute exercise triggered increases in circulating free fatty acids and lactate levels both at baseline and after the intervention ($P < 0.05$). Noteworthy, acute exercise following training resulted in a 71% increase in SWS during subsequent sleep in comparison with the nonexercise condition before training, respectively 2.4% and 1.4% ($P < 0.05$). Following training, acute exercise reduced total wake time by 30% and REM onset latency by 14% ($P < 0.05$). Acute exercise improved sleep continuity by decreasing total wake time. These results show that aerobic training could increase sleep depth and continuity, during active days, in elderly men. In habitual exercisers, these effects of aerobic exercise on sleep, although modest, might counteract those resulting from aging.



## 1. Introduction

The reduced capacity to initiate and maintain sleep with aging causes a progressive decrease in sleep quality. In fact, the prevalence of sleep disorders is more elevated in seniors [1] and, despite spending more time in bed, older people obtain less (and report more complaints about) sleep [2]. The most consistent age-related changes include an increased fragmentation of sleep and losses in slow-wave sleep (SWS, stages 3 + 4 or deep sleep) [1], the latter being known for its critical restorative function [3]. Both the amplitude (75 μV+) and frequency (0.5–2.0 Hz) of delta waves are reduced [2], decreasing the electroencephalographic (EEG) power spectrum, the so-called 'flattening of the EEG' in the aged. Deleterious clinical outcomes might ensue, such as excessive daytime sleepiness, propensity to fall asleep at the wheel, mood impairments, or metabolic syndrome. On the other hand, the use of sleeping pills in seniors is complicated by possible interactions with other drugs and low tolerance to side effects and is not recommended for long-term use because of its association with excess mortality [4]. Hence, non-pharmacological strategies aimed at promoting sleep are especially indicated in older individuals.

While epidemiological surveys have unveiled that poor exercise habits might contribute to incident insomnia in later life [5], exercise

* Corresponding author at: Research Center on Aging, Health and Social Services Centre—University Institute of Geriatrics of Sherbrooke, Sherbrooke, QC J1H 4C4, Canada.
*E-mail address:* Dominique.Lorrain@USherbrooke.ca (D. Lorrain).

http://dx.doi.org/10.1016/j.physbeh.2014.12.031
0031-9384/

has proven to offer a number of positive effects on sleep, including total sleep time (TST), sleep onset latency, SWS, sleep efficiency, sleep quality, self-rated time to fall asleep, and nocturia [6–8]. However, many studies have relied on questionnaires. Although self-reported sleep ratings are somewhat costless and allow the appraisal of a number of variables, their subjectiveness might be misleading, i.e. subject to bias in report and/or recall. Prior studies that used polysomnography (PSG) to investigate the effect of exercise training on sleep in seniors have been limited in number. Among these, one could find a study involving subjects presenting moderate sleep complaints [9] and two reports that considered as 'older' individuals who were actually in their early forties [10,11]. However, a home-based PSG study revealed that aerobically trained older men had more SWS than untrained controls, irrespective of whether sleep followed an active or inactive day [12]. However, because of the cross-sectional nature of this study, it cannot be ruled out that genetic endowments, aerobic training or other factors were involved. For example, good sleepers might be more prone to adhere to exercise training, which could reciprocally cause benefits to their sleep.

In keeping with the aforementioned studies, one could note that none used controlled, laboratory-based PSG combined with an exercise intervention to examine the effect of aerobic training on sleep in older adults. Therefore, the current study aimed to investigate the effects of aerobic exercise training on sleep depth and continuity in older men.

## 2. Methods

### 2.1. Subjects

Thirteen community-dwelling Caucasian men aged 64 ± 3, range 57–70 yrs were recruited using local advertisement. Subjects had no orthopedic limitations, were not engaged in regular exercise training, and did not take any medication acting on sleep or serotonergic tone, e.g. tricyclic antidepressants, antipsychotics, or monoamine oxidase inhibitors during the year prior to the study. None had diabetes mellitus, obesity or smoked. In addition, the following exclusion criteria were applied on PSG data of the first night: leg movement index > 15 $h^{-1}$ causing arousals or wake [13]; more than 10 $h^{-1}$ episodes of sleep apnea and/or hypopnea (respectively >80% and >50% reduction of airflow mean amplitude) causing arousals or wake; professional evening or night activities; regular nap habits (>30 min); or a Pittsburgh Sleep Quality Index > 5. The Human Research Ethics Committee of the University Institute of Geriatrics of Sherbrooke approved this study and subjects were informed about the risks and benefits of the study before providing written consent. One subject was excluded from analyses on the basis of the Obstructive Sleep Apnea–Hypopnea Index criterion.

### 2.2. General study design

Volunteers were screened for eligibility by a phone interview, then eligible subjects were invited for further study explanations and written consent. At first, subjects underwent a maximal, pretesting cardiovascular examination consisting of a graded cardiopulmonary test to volitional exhaustion which aimed to assess ventilatory threshold, respiratory compensation threshold, maximal heart rate and peak $O_2$ consumption ($\dot{V}O_2$ peak). Criteria for determination of $\dot{V}O_2$ peak were attainment of maximal age-predicted heart rate, no further increase in $\dot{V}O_2$ despite increased workload, or subject's desire to stop. Then, subjects underwent a body composition scan, after which sleep was recorded at the laboratory over the course of 3 consecutive nights: 1) familiarization night, 2) nonexercise sedentary trial (SED), and 3) exercise trial (EXR). Regarding pre- and post-training experimental exercise challenges, subjects first warmed-up for 5–10 min, and then exercise at a moderate intensity for 1 h. This was immediately followed by a self-controlled recovery period (~5 min). During both exercise challenges, blood sampling analyses were conducted (before, during, and after exercise) in the fasted state to confirm whether exercise elicited a physiological challenge at baseline and following training. Hence, free fatty acids levels were taken as an index of availability of circulating energy substrates, whereas lactate served as an index of 'anaerobic' glycolysis [14]. The aforementioned tests were repeated following training for comparison purposes.

### 2.3. Maximal cardiorespiratory testing

Subjects got acquainted with cardiopulmonary testing before their participation in the study. The Physical Activity Readiness Questionnaire was first filled, and then subjects engaged in a maximal, modified Balke treadmill test in which blood pressure and electrocardiogram were interpreted by a physician at each exercise level. Oxygen uptake was measured from gas exchange every 5 s using an automated Oxycon Pro System (Jaeger; Würzburg, Germany) calibrated with reference gases before each test. $\dot{V}O_2$ peak was defined as the highest $\dot{V}O_2$ value observed in the last minute of the test.

### 2.4. Threshold determination

The ventilatory threshold was identified using the inflection point in the ventilatory equivalent for $O_2$ vs. $\dot{V}O_2$ curve with no concomitant rise in $\dot{V}E/\dot{V}CO_2$ [15]; respiratory compensation was identified using the loss of linearity of the ventilatory equivalent for $CO_2$ vs. $\dot{V}O_2$ curve [16]. Training-induced changes in the ventilatory- and respiratory compensation thresholds served as indices of improvements in cardiorespiratory fitness.

### 2.5. Body composition

Fat mass and lean body mass were determined using dual-energy X-ray absorptiometry (DXA Prodigy; Lunar Corp., Madison, WI, USA). The coefficients of variation for repeated determination of fat mass and fat-free mass in a subgroup of 10 individuals were 4.7% and 1.1%, respectively.

### 2.6. Experimental exercise sessions

The pre- and post-training exercise challenges consisted in 1 h of steady-state, incline treadmill brisk walking (~68–69% $\dot{V}O_2$ peak), followed by 30 min of rest. The target zone (i.e. reached in ~5–10 min). Exercise was performed before noon after a 3-h fast or more. Practically, subjects brisk walked at ~6 km $h^{-1}$ with the grade of the treadmill adjusted between 4 and 12% depending on fitness.

### 2.7. Training program

Aerobic exercise sessions were supervised and held thrice weekly (non-consecutive days) for 16 wks with no interruption. Missed sessions were re-scheduled when possible. Exercise consisted of 45 min of brisk walking at ~6 km $h^{-1}$ on an inclined treadmill adjusted to individual fitness. Exercise intensity was monitored using a telemetric heart rate monitor (Polar Electro Canada Inc., Lachine, QC, Canada) using heart rate at ventilatory threshold (lower bound) and respiratory compensation threshold (upper bound) obtained during the maximal cardiopulmonary test. Five to 10 min was allowed to warm-up and cool-down (self-paced), and training intensity was progressively increased over the first training sessions.

### 2.8. Blood analyses

Whole blood was immediately centrifuged (3000 rpm × 15 min) and stored at −82 °C. For analysis of lactate (lactic dehydrogenase

method) and free fatty acids (enzymatic colorimetric method), plasma samples were transferred into autosampler vials and run in duplicate using an automated clinical chemistry system (Dimension® XP and Plus; Dade Behring, Newark, DE). The intra-assay coefficients of variation for duplicate measures were of 2.1% for lactate ($n = 66$) and 2.0% for free fatty acids ($n = 101$).

### 2.9. Sleep electrophysiology

Before and after the intervention, the 1st night of each 3-night sequence served as a familiarization night (data discarded from analyses). The 2nd night was recorded after a nonexercise SED day before training, in which subjects were instructed to maintain physical activity low; data of the 3rd night were obtained after an EXR day. The 2nd night served as the control night (CON). Subjects were instructed to avoid daytime napping, to maintain their customary bedtime hours, to refrain from alcohol and caffeine during the 24-h period before each PSG record, and to avoid napping on recording days. Eight hours of sleep were recorded in each trial from 21:00–23:30 p.m. (lights off) to 05:00–07:30 a.m. (lights on). Electric signals were amplified using a 13-channel PSG setting (Model 12 Amplifier System, Grass Medical Instruments), sampled at a frequency window of 0.3–100 Hz per channel, and stored on computer disk for later analysis using a commercial software (Harmonie, Stellate Systems, Montreal, QC, Canada). Electrodes were installed by qualified medical electrophysiology technicians, then PSG records were obtained using combined EEG ($F_3$-$A_2$, $F_4$-$A_1$, $C_3$-$A_2$, $C_4$-$A_1$, $O_1$-$A_2$, $O_2$-$A_1$, Cz-$A_2$, Pz-$A_2$, Fz-$A_2$) using electromyography (EMG) (chin and *M. tibialis anterior*), electro-oculography (EOG, left and right), and electrocardiography ($V_2$ lead). Breathing was monitored using an orobuccal thermistor. Sleep stages were scored in 30-s epochs by two raters according to standard criteria [17]. The inter-rater epoch-to-epoch coefficient of variation (after the 2nd rater selected at random and rated sleep samples in all subjects) was below 5%.

The following variables were obtained: total recording time (time elapsed between 'lights off' and 'lights on'); TST; sleep efficiency (TST × 100 / total min in bed with 'lights off'); wake after sleep onset; stage-1 sleep; stage-2 sleep; SWS (stages 3 + 4 or delta sleep; waves of 0.5–2.0 Hz and ≥75 μV from peak to peak); sleep onset latency, time elapsed between lights off until the onset of at least 60 s of stage-1 sleep or stage-2 onset; rapid eye movement (REM); non-REM sleep (NREM) (stages 2, 4); REM onset latency; wake number; and total wake time. Leg movements were identified as leg EMG activity (*M. tibialis anterior*) at least 125% that at baseline, lasting from 0.5 to 5 s, with arousal following leg movements by no more than 3 s [13]. A visual inspection was conducted following automated analysis to confirm leg movements. Apnea and hypopnea episodes were taken as >80% and >50% reductions of airflow mean amplitude causing arousals or wake. Arousals were defined as shifts in EEG sleep frequency for 3 s or more. The arousal index was defined as the number of arousals per hour of sleep.

### 2.10. Sleep—self-reported ratings

The Pittsburgh Sleep Quality Index was filled at baseline and follow-up to obtain a subjective sleep evaluation. From 10 questions, 7 component scores (sleep duration, sleep disturbance, sleep latency, day dysfunction, sleep efficiency, overall sleep quality, and sleep medications; range, 0–3) as well as a global score (range, 0–21) were obtained.

### 2.11. Analyses

Data are presented as means ± standard deviations (SD) unless otherwise specified. The Kolmogorov–Smirnov statistic served to test normality of distribution of data. Two-tailed paired Student *t*-tests were used to detect differences between subject characteristics and blood data between baseline and follow-up; the Wilcoxon signed rank test served for data that did not show a normal distribution. One-way repeated measures ANOVA served to detect differences between sleep variables; Dunnett *post-hoc* tests served to specify differences between means. For data showing a non-Gaussian distribution, the Friedman statistic with Dunn's *post-hoc* tests were applied. For blood variables, given that some subjects had one missing value during serial sampling, multiple paired *t*-tests were used to detect differences between means. The threshold to declare significance was thus set at $P < 0.017$ using the standard Bonferroni adjustment for multiple comparisons (recalculated $\alpha = 0.05/3$). The main effects of training, acute exercise, and their interaction, were assessed using 2-way repeated measures ANOVA [training factor (baseline vs. post-training) X activity factor (SED vs. EXR)]. The Wilcoxon signed rank test served to test for differences between pre and post-training subjective ratings. Significance was declared at $P \leq 0.05$ (except for blood variables, see above). Analyses were run using the GraphPad Prism software (Version 5.04; GraphPad Software, Inc., San Diego, CA, USA).

## 3. Results

### 3.1. Subject characteristics

Thirteen men aged 64 ± 3, range 57–70 yrs completed the physical training program (Table 1). Missed exercise sessions were rescheduled when possible, resulting in an attendance rate of 97% over 16 wks. Improvements in some components of aerobic fitness were noted in response to training, that is, ventilatory threshold showed an increase of 8.2% ($P < 0.05$) and respiratory compensation threshold was increased by 6.6% ($P < 0.05$).

### 3.2. Metabolic effects of exercise

Resting blood lactate level rose in response to acute exercise both before (1.1 ± 0.3 to 4.1 ± 2.1 mmol $L^{-1}$, $P < 0.017$) and following aerobic training (0.9 ± 0.4 to 1.9 ± 0.6 mmol $L^{-1}$, $P < 0.001$). Acute exercise also increased resting free fatty acid levels at baseline (0.42 ± 0.18 to 1.03 ± 0.36 mmol $L^{-1}$, $P < 0.001$) and following training (0.30 ± 0.15 to 0.75 ± 0.32 mmol $L^{-1}$, $P < 0.001$) in the older men.

### 3.3. Sleep electrophysiology

Sleep efficiency was of 84% in CON and of 87% for both baseline and post-training EXR conditions (Table 2). Baseline sleep efficiency tended to differ between EXR and CON conditions ($P = 0.076$) as well as post-training-EXR and CON ($P = 0.076$). As illustrated in Fig. 1, analyses revealed that EXR following training was followed by a higher amount

**Table 1**
Characteristics of subjects before and after aerobic exercise training.

| Variable | Training, 16 wks | | |
|---|---|---|---|
| | Baseline | Post-training | Diff. (%) |
| Age (yrs) | 64 ± 3 | – | ~ |
| Height (m) | 1.77 ± 0.05 | – | ~ |
| Weight (kg) | 84.9 ± 11.3 | 84.0 ± 11.0 | (−1.0) |
| BMI ($kg \cdot m^{-2}$) | 27.1 ± 2.7 | 26.8 ± 2.7 | (−1.1) |
| Body fat (%) | 25.6 ± 6.9 | 25.2 ± 7.5 | (−1.6) |
| Fat-free mass (%) | 71.7 ± 6.6 | 71.9 ± 7.2 | (+0.3) |
| $\dot{V}O_2$ peak ($mL \cdot kg^{-1} \cdot min^{-1}$) | 36.5 ± 6.7 | 38.2 ± 6.5 | (+4.6) |
| Ventilatory threshold ($mL \cdot kg^{-1} \cdot min^{-1}$) | 18.2 ± 2.7 | 19.7 ± 3.3* | (+8.2) |
| RC ($mL \cdot kg^{-1} \cdot min^{-1}$) | 27.2 ± 4.7 | 29.0 ± 5.0* | (+6.6) |
| Maximal heart rate (bpm) | 154 ± 13 | 154 ± 8 | – |

Abbreviations: BMI, body mass index; $\dot{V}O_2$ peak, peak $O_2$ consumption; RC, respiratory compensation threshold.
Values are means ± SD from 13 subjects.
Superscripts denote statistical difference vs. pre-training.
* $P < 0.05$.

**Table 2**
The effect of acute exercise on sleep continuity and architecture before and after 16 wks of training in older men.

| Variable | Pre-training | | Post-training | | *P*-value* |
|---|---|---|---|---|---|
| | SED | EXR | SED | EXR | 2-way ANOVA |
| Total recording time (min) | 485 ± 1 | 486 ± 1 | 486 ± 1 | 485 ± 0 | $P > 0.05$ |
| Sleep efficiency (%) | 83.9 ± 2.0 | 86.9 ± 1.8 | 82.6 ± 1.9 | 86.9 ± 1.5 | $P = 0.08$ |
| Total sleep time (min) | 407 ± 10 | 422 ± 9 | 402 ± 9 | 422 ± 8 | $P = 0.08$ |
| Sleep onset latency (min) | 3.9 ± 0.6 | 3.3 ± 0.5 | 4.5 ± 1.4 | 4.6 ± 1.8 | $P > 0.05$ |
| Stage 1 (min) | 47.6 ± 3.4 | 38.8 ± 3.7 | 47.6 ± 4.2 | 46.3 ± 3.6 | $P > 0.05$ |
| % TST | 11.8 ± 0.9 | 9.3 ± 0.9 | 11.9 ± 1.1 | 11.1 ± 1.0 | $P > 0.05$ |
| Stage 2 (min) | 273.8 ± 10.0 | 296.2 ± 8.3 | 269.0 ± 8.8 | 278.7 ± 7.3 | $P = 0.08$ |
| Slow-wave sleep (min) | 5.5 ± 2.2 | 7.8 ± 2.8 | 6.8 ± 2.5 | 10.5 ± 3.5 | $P = 0.07$ |
| REM (min) | 80.2 ± 5.5 | 79.6 ± 7.5 | 78.4 ± 4.6 | 86.5 ± 5.3 | $P > 0.05$ |
| %TST | 19.7 ± 1.2 | 18.6 ± 1.6 | 19.5 ± 1.1 | 20.4 ± 1.1 | $P > 0.05$ |
| Wake number (*n*) | 13 ± 2 | 9 ± 1 | 10 ± 1 | 11 ± 2 | $P > 0.05$ |
| Total wake time (min) | 59.8 ± 9.4 | 41.7 ± 8.6 | 64.4 ± 9.0 | 45.4 ± 6.6 | $P = 0.02$ |

EXR, exercise condition; NREM, non-REM (stages 2–4); REM, rapid eye movements sleep; SED, sedentary condition; TST, total sleep time.
Values are means ± SE ($n = 13$).
* Significance of main effects, 2-way repeated-measures ANOVA.

of SWS (+71%) during subsequent sleep compared with CON, i.e. respectively 2.4 ± 0.8% and 1.4 ± 0.5% ($P < 0.05$). Differences were observed for NREM sleep ($P < 0.05$); post-hoc analyses revealed that NREM sleep was higher in baseline EXR compared with CON (respectively 304 ± 8 vs. 279 ± 10 min; $P < 0.05$). As shown in Fig. 2, there was a significant main effect of aerobic training on wake after sleep onset ($P = 0.01$), the raw values were of 64.5 min in CON and of 46.5 min in post-training EXR. Sleep onset latency was below 5 min in all trials. Absolute values of stage-2 sleep differed between conditions ($P < 0.05$), although they only reached a trend when expressed as % TST ($P = 0.063$).

### 3.4. Self-reported sleep ratings

The Pittsburgh Sleep Quality Index did not allow detecting any changes in sleep in response to aerobic training. Selected component scores at baseline and post-training, respectively, were as follows: sleep quality, 0.54 ± 0.52 and 0.62 ± 0.51 ($P > 0.05$); sleep latency, 0.31 ± 0.63 and 0.31 ± 0.63 ($P > 0.05$); sleep duration, 0.46 ± 0.52 and 0.46 ± 0.66 ($P > 0.05$); sleep efficiency, 0.31 ± 0.63 and 0.62 ± 0.77 ($P > 0.05$); and sleep disturbances, 1.31 ± 0.48 and 1.23 ± 0.44 ($P > 0.05$).

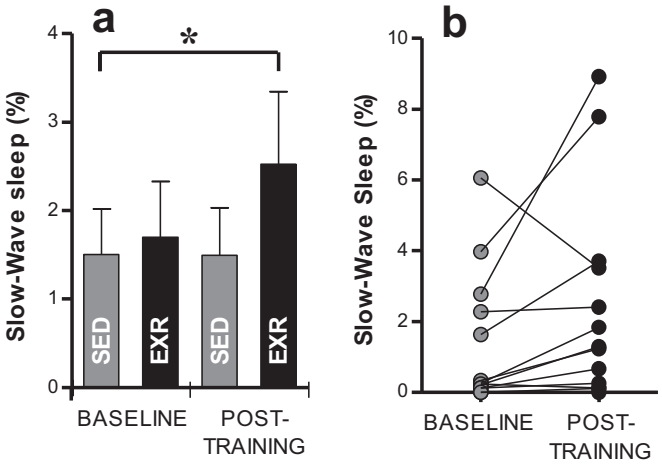


**Fig. 1.** (a) Impact of 1 h of moderate exercise on slow-wave sleep (SWS) at 0 and 16 wks of aerobic training in older men. Values are means ± SE; results from one-way repeated-measures ANOVA ($P < 0.05$). (b) Line graph of individual SWS data before and after training. Sleep was recorded at baseline after a day containing no exercise (left) and after training during a night subsequent to exercise (right). EXR, exercise condition; SED, sedentary condition ($n = 13$).

## 4. Discussion

The primary aim of this study was to investigate the effect of aerobic exercise training on sleep depth and continuity in older adults. Few studies have compared objective sleep data gathered before and after controlled, supervised aerobic training in seniors. The present results show that acute exercise (1 h of moderate-intensity exercise) performed after 16 wks of training led to an increased amount of SWS during subsequent sleep in comparison with a sedentary day before training. These results strengthen independent observations in seniors [12] and are in agreement with the premise that 'fitness facilitates sleep' [18] since we could only find differences in SWS when pre- and post-training values were compared (exercise vs. nonexercise conditions). Exercise also improved sleep consolidation by reducing wake time, which strengthens earlier observations gathered in the elderly [19]. The older men spent an average of 1.4% of nighttime in SWS following the inactive trial against 2.4% in the post-training exercise trial (relative change, +71%). Given that SWS shows a progressive reduction as one ages, it could be possible that a floor effect helped to find differences in this variable. Regarding the nonexercise condition before and after training, we found a similar proportion of SWS, which corroborates previous findings [20]. This suggests that the physiological stress elicited by exercise, rather than aerobic training, is key to influence SWS. Given that our subjects increased their absolute intensity of exercise following training, the additional work involved in post- vs. pre-training exercise in a time window of 1 h might help explain why SWS levels differed between nights 2 and 5 but not between nights 2 and 3. Previous attention has been paid regarding the exercise-induced changes in adrenocortical activity, i.e. index of physiological stress, during aerobic exercise at 35% $\dot{V}O_2$ max and their effect on sleep depth in trained men [21]. In that study, it is worth noting that subjects in whom adrenergic activity increased did not exhibit higher SWS values during subsequent sleep, whereas those with unchanged activity exhibited increased SWS [21]. One could note that the aftermath of exercise on deep sleep could occur beyond the 1st night [3]. In the current study, we have monitored 2 physiological markers of the adrenergic stress induced by exercise, i.e. found increased circulating lactate and free fatty acid levels.

It is believed that neurons involved in the onset of sleep lie in the brain's preoptic region [22] and might be influenced by arousal systems via transmitters of wakefulness, e.g. noradrenaline, serotonin, glutamate, acetylcholine [23]. The Raphe nuclei send serotonin-containing projections to the forebrain whose discharge is maximal during wake [23], and who owns biphasic effects on sleep, i.e. promote wake and then sleep. On the other hand, it is interesting that dozens of studies found sizeable effects of tryptophan administration (the only serotonin precursor) on sleep, e.g. reductions in sleep onset latency [24].

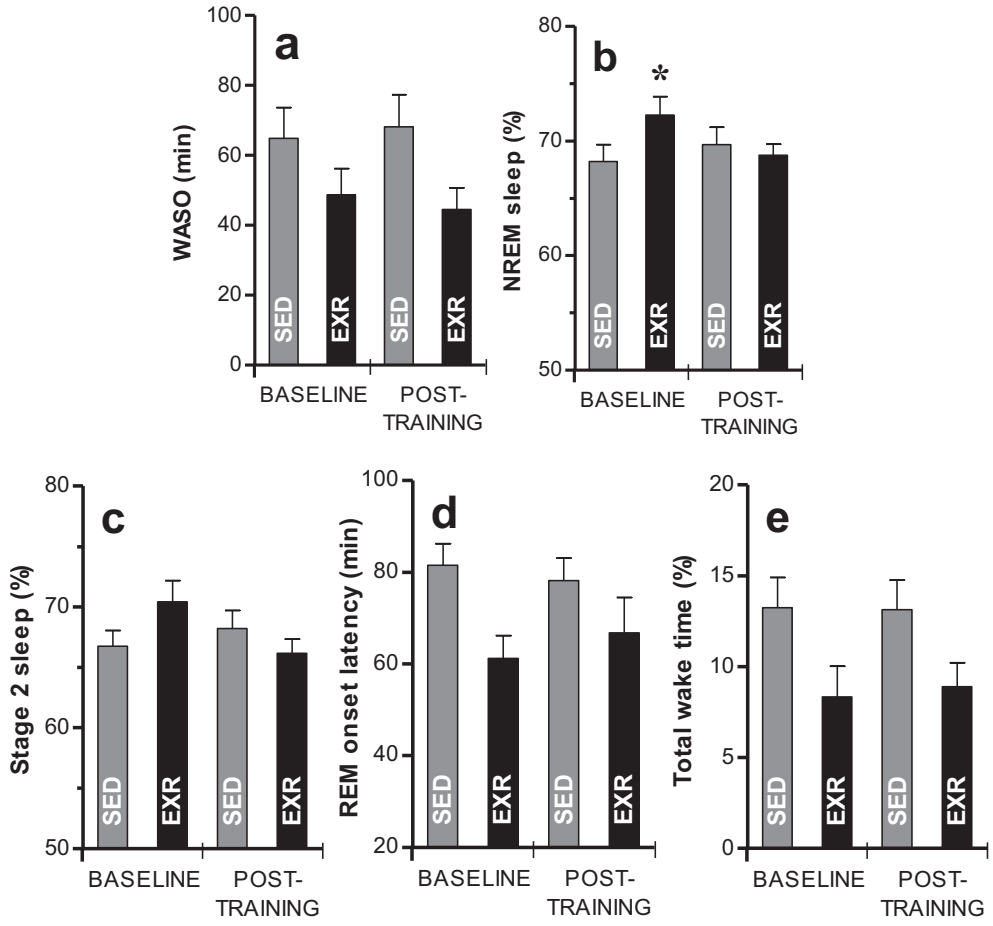


**Fig. 2.** Effects of acute exercise before and after 16 wks of aerobic training on wake after sleep onset (a), non-REM sleep (b), stage 2 sleep (c), REM onset latency (d), and total wake time (e) in older men. Analyses revealed significant main effects of exercise on wake after sleep onset ($P = 0.046$), REM onset latency ($P = 0.018$), and total wake time ($P = 0.019$) as well as an interaction between exercise and training for stage 2 sleep ($P = 0.002$) and non-REM ($P = 0.006$). SED, sedentary condition; EXR, exercise condition; WASO, wake after sleep onset; non-REM, non-rapid eyes movement sleep (stages 2–4). Values are means ± SE. *Different from baseline/SED ($P < 0.05$). $n = 13$.

Therefore, if one considers that exercise could increase tryptophan availability to the brain [25], then increases in serotonergic activity associated with exercise might possibly underlie, in part, the effects of exercise on sleep. However, we are unaware of any evidence in support of this assumption.

According to the homeostatic theory of sleep, the activity–rest cycle involves an equilibrium between two opposing phenomena, where any elevation in neural activity is compensated by proportional increases in SWS [26]. It was recently demonstrated that immobilizing an arm during the day could elicit significant reductions in slow-wave activity over the same brain area during subsequent sleep [27]. If one considers that the primary motor cortex (located in the more anterior part of the cortex) is strongly activated during voluntary movements [26] and that delta waves could most easily be detected in the more anterior derivations [28], then one might speculate that the elevated activity of the motor cortex associated with exercise could be linked with the compensatory increase in SWS found after physical exercise.

Some investigators used both objective and subjective measures to assess the effects of exercise on sleep in the elderly (PSG and Pittsburgh Sleep Quality Index, respectively) and could detect sizeable effects only via subjective means [29]. This contrasts with the present results because we found changes only through PSG means. However, because the Pittsburgh Sleep Quality Index involves a 30-day assessment of sleep (which would imply *both* EXR and SED conditions here), it is thus of no surprise that we could not find significant differences using this questionnaire. It should be outlined that the sample size of the present study was modest ($n = 13$), so that it could not be ruled out that a larger sample could have led to detect additional changes in this population. Lastly, the present older men did not report any sleep complaints before the study; it is then felt that the effect of exercise training on sleep should be equal or larger in a clinical population. However, given that sleep quality and quantity are both adversely affected with age, interventions aimed at promoting sleep deserve attention in this population.

## 5. Conclusions

The present results show that 1 h of moderate-intensity aerobic exercise performed following aerobic training increases subsequent sleep depth and continuity in older men, respectively by increasing %SWS and decreasing total wake time. Our findings indicate that daily repeated bouts of exercise might be beneficial for sleep depth and continuity in healthy older men. The effects of chronic exercise run counter to those occurring with normal aging and might ultimately offer an alternative strategy to help deepen and consolidate sleep in older individuals.

## Conflict of interest

Each author has nothing to declare.

## Acknowledgments

This work was funded by a grant from the Research Centre on Aging, Health and Social Services Centre—University Institute of Geriatrics of

Sherbrooke. M.O.M. was supported by the *Réseau de formation en recherche sur la santé et vieillissement*. I.J.D. received a salary grant from the *Fonds de recherche du Québec — santé* (FRQS). The authors thank the subjects for their participation in the study, and M. Fisch, R.N., for blood withdrawals.

## References

[1] D.L. Bliwise, Normal aging, in: M.H. Kryger, T. Roth, W.H. Dement (Eds.), Principles and Practice of Sleep Medicine, Saunders, Philadelphia, 2000, pp. 26–42.
[2] I. Feinberg, R.L. Koresko, N. Heller, EEG sleep patterns as a function of normal and pathological aging in man, J. Psychiatr. Res. 5 (2) (1967) 107–144.
[3] C.M. Shapiro, R. Bortz, D. Mitchell, P. Bartel, P. Jooste, Slow-wave sleep: a recovery period after exercise, Science 214 (4526) (1981) 1253–1254.
[4] G. Belleville, Mortality hazard associated with anxiolytic and hypnotic drug use in the National Population Health Survey, Can. J. Psychiatry 55 (9) (2010) 558–567.
[5] K. Morgan, Daytime activity and risk factors for late-life insomnia, J. Sleep Res. 12 (3) (2003) 231–238.
[6] P. Montgomery, J. Dennis, Physical exercise for sleep problems in adults aged 60+, Cochrane Database Syst. Rev. 4 (2002) CD003404.
[7] J.M. Dzierzewski, M.P. Buman, P.R. Giacobbi Jr., B.L. Roberts, A.T. Aiken-Morgan, M. Marsiske, et al., Exercise and sleep in community-dwelling older adults: evidence for a reciprocal relationship, J. Sleep Res. 23 (1) (2014) 61–68.
[8] M.P. Buman, E.B. Hekler, D.L. Bliwise, A.C. King, Exercise effects on night-to-night fluctuations in self-rated sleep among older adults with sleep complaints, J. Sleep Res. 20 (1) (2011) 28–37.
[9] A.C. King, R.F. Oman, G.S. Brassington, D.L. Bliwise, W.L. Haskell, Moderate-intensity exercise and self-rated quality of sleep in older adults. A randomized controlled trial, JAMA 277 (1) (1997) 32–37.
[10] I. Montgomery, J. Trinder, S. Paxton, D. Harris, G. Fraser, I. Colrain, Physical exercise and sleep: the effect of the age and sex of the subjects and type of exercise, Acta Physiol. Scand. Suppl. 574 (1988) 36–40.
[11] I.J. Montgomery, J. Trinder, G. Fraser, S.J. Paxton, Aerobic fitness and exercise: effect on the sleep of younger and older adults, Aust. J. Psychol. 39 (1987) 259–272 (1987).
[12] J.D. Edinger, M.C. Morey, R.J. Sullivan, M.B. Higginbotham, G.R. Marsh, D.S. Dailey, et al., Aerobic fitness, acute exercise and sleep in older men, Sleep 16 (4) (1993) 351–359.
[13] M. Bonnet, D. Carley, M. Carskadon, P. Easton, C. Guilleminault, R. Harper, et al., ASDA report. Recording and scoring leg movements, Sleep 16 (8) (1993) 748–759.
[14] A. Katz, K. Sahlin, Regulation of lactic acid production during exercise, J. Appl. Physiol. 65 (2) (1988) 509–518.
[15] V.J. Caiozzo, J.A. Davis, J.F. Ellis, J.L. Azus, R. Vandagriff, C.A. Prietto, et al., A comparison of gas exchange indices used to detect the anaerobic threshold, J. Appl. Physiol. 53 (5) (1982) 1184–1189.
[16] W.L. Beaver, K. Wasserman, B.J. Whipp, A new method for detecting anaerobic threshold by gas exchange, J. Appl. Physiol. 60 (6) (1986) 2020–2027.
[17] A. Rechtschaffen, A. Kales, A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects, U.S. Public Health Service, U.S. Government Printing Office, Washington D.C., 1968
[18] C.M. Shapiro, P.M. Warren, J. Trinder, S.J. Paxton, I. Oswald, D.C. Flenley, et al., Fitness facilitates sleep, Eur. J. Appl. Physiol. Occup. Physiol. 53 (1) (1984) 1–4.
[19] F.S. Lira, G.D. Pimentel, R.V. Santos, L.M. Oyama, A.R. Damaso, C.M. Oller do Nascimento, et al., Exercise training improves sleep pattern and metabolic profile in elderly people in a time-dependent manner, Lipids Health Dis. 10 (2011) 1–6.
[20] H.S. Driver, A.F. Meintjes, G.G. Rogers, C.M. Shapiro, Submaximal exercise effects on sleep patterns in young women before and after an aerobic training programme, Acta Physiol. Scand. Suppl. 574 (1988) 8–13.
[21] A. Buguet, B. Roussel, R. Angus, B. Sabiston, M. Radomski, Human sleep and adrenal individual reactions to exercise, Electroencephalogr. Clin. Neurophysiol. 49 (5–6) (1980) 515–523.
[22] J.E. Sherin, P.J. Shiromani, R.W. McCarley, C.B. Saper, Activation of ventrolateral preoptic neurons during sleep, Science 271 (5246) (1996) 216–219.
[23] B.E. Jones, From waking to sleeping: neuronal and chemical substrates, Trends Pharmacol. Sci. 26 (11) (Nov 2005) 578–586.
[24] E. Hartmann, D. Greenwald, Tryptophan and human sleep: an analysis of 43 studies, in: H.G. Schlossberger, W. Kochen, B. Linzen, H. Steinhart (Eds.), Progress in Tryptophan and Serotonin Research, Walter de Gruyter, Berlin, 1984, pp. 297–304.
[25] M.O. Melancon, D. Lorrain, I.J. Dionne, Exercise increases tryptophan availability to the brain in older men aged 57–70 years, Med. Sci. Sports Exerc. 44 (5) (2012) 881–887.
[26] T.H. Petersen, M. Willerslev-Olsen, B.A. Conway, J.B. Nielsen, The motor cortex drives the muscles during walking in human subjects, J. Physiol. 590 (10) (2012) 2443–2452.
[27] R. Huber, M.F. Ghilardi, M. Massimini, F. Ferrarelli, B.A. Riedner, M.J. Peterson, et al., Arm immobilization causes cortical plastic changes and locally decreases sleep slow wave activity, Nat. Neurosci. 9 (9) (2006) 1169–1176.
[28] M. Munch, V. Knoblauch, K. Blatter, C. Schroder, C. Schnitzler, K. Krauchi, et al., The frontal predominance in human EEG delta activity after sleep loss decreases with age, Eur. J. Neurosci. 20 (5) (2004) 1402–1410.
[29] S. Benloucif, L. Orbeta, R. Ortiz, I. Janssen, S.I. Finkel, J. Bleiberg, et al., Morning or evening activity improves neuropsychological performance and subjective sleep quality in older adults, Sleep 27 (8) (2004) 1542–1551.